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**Literature search results**

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**Search details**

Paediatric asthma patients and exposure to passive smoking. Are there any studies which show passive smoking as a trigger for significant asthma?

**Resources searched**

NHS Evidence; TRIP Database; Cochrane Library; CINAHL; EMBASE; MEDLINE; Google Scholar

**Database search terms**: asthma*; ASThma; paediatric*; pediatric*; exp CHILD; exp INFANT; chil*; infant*; "young person*"; "young people"; adolescent*; ADOLESCENT; youth*; teenager*; "passive smok*"; TOBACCO SMIKE POLLUTION; secondhand adj2 smok*; second-hand adj2 smok*; "second hand" adj2 smok*; passiv* adj2 smok*; environmental tobacco smok*; DEATH; death; dying; fatal* adj2 outcome*; FATAL OUTCOME; life adj2 threaten*; acute adj1 severe; exacerbation*; trigger*; severe; severity; acute; SEVERITY OF ILLNESS; ACUTE DISEASE; CATASTROPHIC ILLNESS; CRITICAL ILLNESS; "systematic" review*; metaanalysis*; meta-analysis*; META-ANALYSIS; "random* controlled" adj0 (trial* OR stud*); RCT*; RANDOMIZED CONTROLLED TRIAL; safeguarding; chil* adj2 protecti*  

**Evidence search string(s)**: asthma ("secondhand smok*" OR "passive smok*" OR "second hand smok*" OR "environmental tobacco smok*") (child* OR infant* OR adolescent* OR youth* OR teenager* OR "young people" OR "young person")  

("passive smoking" OR "secondhand smoke" OR ETS) asthma (safeguarding OR "child protection")

**Google search string(s)**: ~asthma (~"secondhand smoke" OR ~"passive smoking" OR ~"environmental tobacco smok") (~child OR ~infant OR ~adolescent OR ~youths OR ~teenager OR ~"young people") (~metaanalysis OR ~"systematic review" OR ~"randomized controlled trial" OR RCT)  

~asthma (~"secondhand smoke" OR ~"passive smoking" OR ~"environmental tobacco
Summary

There is evidence that second-hand or passive smoke has an effect on asthma in children; however most of it seems not to have been published recently, or not as a systematic review or RCT. If you want me to extend the search to cover other types of study, or earlier research, please let me know.

Guidelines

Royal College of Physicians

Passive smoking and children: a report by the Tobacco Advisory Group of the Royal College of Physicians 2010

1. Estimated effects of passive smoking and asthma occurring for the first time in the various age ranges are shown in Table 4.2. With the exception of prenatal maternal smoking, which had a relatively strong effect on asthma occurring in the first two years (OR 1.91, 95% CI 1.43 to 2.53, four studies), the effects of passive smoke exposure on asthma were less strong than those on wheeze, with the strongest effect (for household smoking on asthma collected after 5 years old) being an OR of 1.50 (95% CI 1.13 to 1.97, four studies). Passive smoking effects were of similar magnitude for asthma at all ages studied. As for wheeze, there was limited information on the relation between paternal smoking and incident asthma. (p. 86)

2. Passive smoking also increases the risk of asthma by about 50% in schoolage children exposed to household smoking. (p. 193)

Evidence-based reviews

The New Zealand Medical Journal

Smoky homes: a review of the exposure and effects of secondhand smoke in New Zealand homes, 2005

SHS exposure for children increases the risk of: asthma exacerbations, lower respiratory illness, lung damage, middle ear disease, behavioural and learning problems, and Sudden Infant Death Syndrome (SIDS).7

Published research

1. Predicting asthma exacerbations in children.

Author(s) Forno E, Celedon JC

Citation: Current Opinion in Pulmonary Medicine, January 2012, vol./is. 18/1(63-9), 1070-5287;1531-6971 (2012 Jan)

Publication Date: January 2012

Abstract: PURPOSE OF REVIEW: This review critically assesses recently published literature on predicting asthma exacerbations in children, while also providing general recommendations for future research in this field.RECENT FINDINGS: Current evidence suggests that every effort should be made to provide optimal treatment to achieve adequate asthma control, as this will significantly reduce the risk of severe disease exacerbations. Children who have had at least one asthma exacerbation in the previous year are at highest risk for subsequent exacerbations, regardless of disease severity and/or control. Although several tools and biomarkers to predict asthma exacerbations have been recently developed, these approaches need further validation and/or have only had partial success in identifying children at risk.SUMMARY: Although considerable progress has
been made, much remains to be done. Future studies should clearly differentiate severe asthma exacerbations due to inadequate asthma control from those occurring in children whose asthma is well controlled, utilize standardized definitions of asthma exacerbations, and use a systematic approach to identify the best predictors after accounting for the multiple dimensions of the problem. Our ability to correctly predict the development of severe asthma exacerbations in an individual child should improve in parallel with increased knowledge and/or understanding of the complex interactions among genetic, environmental (e.g. viral infections) and lifestyle (e.g. adherence to treatment) factors underlying these events.

Source: Medline

2. Non-cancer effects of chemical agents on children’s health.

Author(s) Roosli M

Citation: Progress in Biophysics & Molecular Biology, December 2011, vol./is. 107/3(315-22), 0079-6107;1873-1732 (2011 Dec)

Publication Date: December 2011

Abstract: This paper provides an overview about the non-cancer health effects for children from relevant chemical agents in our environment. In addition, a meta-analysis was conducted on the association between sudden infant death syndrome (SIDS) and maternal smoking during pregnancy as well as postnatal exposure to environmental tobacco smoke (ETS). In children, birth deformities, neurodevelopment, reproductive outcomes and respiratory system are mainly affected by chemical exposures. According to recent systematic reviews, evidence is sufficient for cognitive impairments caused by low lead exposure levels. Evidence for neurotoxicity from prenatal methylmercury exposure is sufficient for high exposure levels and limited for low levels. Prenatal exposure to polychlorinated biphenyls (PCB) and related toxicants results in cognitive and motor deficits. Maternal smoking during pregnancy is a risk factor for preterm birth, foetal growth deficit and SIDS. The meta-analytic pooled risk estimate for SIDS based on 15 studies is 2.94 (95% confidence interval: 2.43-3.57). Postnatal exposure to ETS was found to increase the SIDS risk by a factor of 1.72 (95% CI: 1.28-2.30) based on six studies which took into account maternal smoking during pregnancy. Additionally, postnatal ETS exposure causes acute respiratory infections, ear problems, respiratory symptoms, more severe asthma, and it slows lung growth. These health effects are also of concern for postnatal exposure to ambient and indoor air pollution. Children differ from adults with respect to several aspects which are relevant for assessing their health risk. Thus, independent evaluation of toxicity in childhood populations is essential. Copyright 2011 Elsevier Ltd. All rights reserved.

Source: Medline

3. A randomized trial of parental behavioral counseling and cotinine feedback for lowering environmental tobacco smoke exposure in children with asthma: results of the LET’S Manage Asthma trial.

Author(s) Wilson SR, Farber HJ, Knowles SB, Lavori PW

Citation: Chest, March 2011, vol./is. 139/3(581-90), 0012-3692;1931-3543 (2011 Mar)

Publication Date: March 2011

Abstract: BACKGROUND: Secondhand tobacco smoke exposure impairs the control of pediatric asthma. Evidence of the efficacy of interventions to reduce children’s exposure and improve disease outcomes has been inconclusive.METHODS: Caregivers of 519 children aged 3 to 12 years with asthma and reported smoke exposure attended two baseline assessment visits, which involved a parent interview, sampling of the children’s urine (for cotinine assay), and spirometry (children>=5 years). The caregivers and children (n=352) with significant documented exposure (cotinine>=10 ng/mL) attended a basic asthma education session, provided a third urine sample, and were randomized to the Lowering Environmental Tobacco Smoke: LET’S Manage Asthma (LET’S) intervention (n=178) or usual care (n=174). LET’S included three in-person, stage-of-change-based counseling sessions plus three follow-up phone calls. Cotinine feedback was given at each
in-person session. Follow-up visits at 6 and 12 months postrandomization repeated the baseline data collection. Multivariate regression analyses estimated the intervention effect on the natural logarithm of the cotinine to creatinine ratio (lnCCR), use of health-care services, and other outcomes.

RESULTS: In the sample overall, the children in the LET'S intervention had lower follow-up lnCCR values compared with the children in usual care, but the group difference was not significant (coefficient=-0.307, P=.064), and there was no group difference in the odds of having one asthma-related medical visit (coefficient=0.035, P=.78). However, children with high-risk asthma had statistically lower follow-up lnCCR values compared with children in usual care (coefficient=-1.068, P=.006).

CONCLUSIONS: The LET'S intervention was not associated with a statistically significant reduction in tobacco smoke exposure or use of health-care services in the sample as a whole. However, it appeared effective in reducing exposure in children at high risk for subsequent exacerbations. Trial registry: ClinicalTrials.gov; No.: NCT00217958; URL: clinicaltrials.gov.

Source: Medline

Available in fulltext at Chest; Notes: Username: ULHTKIS/Password: Library


**Author(s)** Treyster Z, Gitterman B

**Citation:** Reviews on Environmental Health, 2011, vol./is. 26/3(187-95), 0048-7554;0048-7554 (2011)

**Publication Date:** 2011

**Abstract:** BACKGROUND: Second hand smoke (SHS) exposure has long been correlated with many adverse disease processes, particularly in children. For children growing up with socioeconomic disadvantages and increased exposure to SHS, exposure can have far-reaching consequences. OBJECTIVE: The purpose of this review was to examine the literature assessing the effects of SHS exposure in children, as well as the perspectives of both parents and providers regarding current practices in cessation counseling. The review also sought out recommendations on ways to increase the influence of pediatricians on parental smoking. STUDY GROUP: Children under the age of 18 years. METHODS: PubMed and MEDLINE were searched systematically. A narrative approach was used because the studies differed in methods and data. RESULTS: The studies showed correlations between SHS exposure and sudden infant death syndrome (SIDS), asthma, altered respiratory function, infection, cardiovascular effects, behavior problems, sleep difficulties, increased cancer risk, and a higher likelihood of smoking initiation. Questionnaires of both parents and pediatricians showed that pediatricians are not consistently carrying out the recommended smoking cessation interventions, with lack of training as a primary barrier. Nevertheless, interventions targeting improved cessation training for both residents and practicing pediatricians have been studied and show promising results. CONCLUSIONS: SHS exposure has many detrimental effects on children’s health, particularly for those in low socioeconomic circumstances, for which factors in the built environment accentuated a higher baseline risk. By counseling parents, expanding residency education, and continuing advocacy work, pediatricians can have a significant positive impact on children's health as related to SHS exposure.

Source: Medline

5. Passive smoking in babies: the BIBE study (Brief Intervention in babies. Effectiveness).


**Citation:** BMC Public Health, 2010, vol./is. 10/(772), 1471-2458;1471-2458 (2010)

**Publication Date:** 2010

**Abstract:** BACKGROUND: There is evidence that exposure to passive smoking in general, and in babies in particular, is an important cause of morbimortality. Passive smoking is
related to an increased risk of pediatric diseases such as sudden death syndrome, acute respiratory diseases, worsening of asthma, acute-chronic middle ear disease and slowing of lung growth. The objective of this article is to describe the BIBE study protocol. The BIBE study aims to determine the effectiveness of a brief intervention within the context of Primary Care, directed to mothers and fathers that smoke, in order to reduce the exposure of babies to passive smoking (ETS). METHODS/DESIGN: Cluster randomized field trial (control and intervention group), multicentric and open. SUBJECT: Fathers and/or mothers who are smokers and their babies (under 18 months) that attend pediatric services in Primary Care in Catalonia. The measurements will be taken at three points in time, in each of the fathers and/or mothers who respond to a questionnaire regarding their baby's clinical background and characteristics of the baby's exposure, together with variables related to the parents' tobacco consumption. A hair sample of the baby will be taken at the beginning of the study and at six months after the initial visit (biological determination of nicotine). The intervention group will apply a brief intervention in passive smoking after specific training and the control group will apply the habitual care. DISCUSSION: Exposure to ETS is an avoidable factor related to infant morbimortality. Interventions to reduce exposure to ETS in babies are potentially beneficial for their health. The BIBE study evaluates an intervention to reduce exposure to ETS that takes advantage of pediatric visits. Interventions in the form of advice, conducted by pediatric professionals, are an excellent opportunity for prevention and protection of infants against the harmful effects of ETS. TRIAL REGISTRATION: ClinicalTrials.gov Identifier: NCT00788996.

Source: Medline
Available in fulltext from BMC Public Health at National Library of Medicine
Available in fulltext from BMC Public Health at EBSCOhost
Available in fulltext from BMC Public Health at BioMedCentral

6. Respiratory diseases related to passive smoking [French] Les pathologies respiratoires liées au tabagisme passif

Author(s) Wirth N., Bohadana A., Spinosa A., Martinet Y.
Citation: Revue Francaise d'Allergologie, November 2009, vol./is. 49/7(534-544), 1877-0320 (November 2009)
Publication Date: November 2009
Abstract: Passive smoking, measured in practice by using specific biomarkers, is a well known factor of morbidity and mortality. The main victims are children, often starting from conception, but adults are not spared. Many respiratory diseases are caused and/or worsened by passive smoking and environmental tobacco smoke (ETS) exposure can have serious health consequences that reduce life expectancy (sudden infant death, respiratory infections, asthma, chronic obstructive pulmonary diseases and lung cancer). Better knowledge of these risks has favourably influenced the legislation banning smoking in enclosed public places in France and in other countries. If one of the main objectives of this measure is to protect non-smokers as well as smokers, its rigorous application fits directly within the goals of prevention and treatment of tobacco dependency. 2009.
Source: EMBASE

7. Epidemiologic evidence of relationships between reproductive and child health outcomes and environmental chemical contaminants.

Author(s) Wigle DT, Arbuckle TE, Turner MC, Berube A, Yang Q, Liu S, Krewski D
Citation: Journal of Toxicology & Environmental Health Part B: Critical Reviews, May 2008, vol./is. 11/5-6(373-517), 1093-7404;1521-6950 (2008 May)
Publication Date: May 2008
Abstract: This review summarizes the level of epidemiologic evidence for relationships between prenatal and/or early life exposure to environmental chemical contaminants and fetal, child, and adult health. Discussion focuses on fetal loss, intrauterine growth restriction, preterm birth, birth defects, respiratory and other childhood diseases,
neuropsychological deficits, premature or delayed sexual maturation, and certain adult cancers linked to fetal or childhood exposures. Environmental exposures considered here include chemical toxicants in air, water, soil/house dust and foods (including human breast milk), and consumer products. Reports reviewed here included original epidemiologic studies (with at least basic descriptions of methods and results), literature reviews, expert group reports, meta-analyses, and pooled analyses. Levels of evidence for causal relationships were categorized as sufficient, limited, or inadequate according to predefined criteria. There was sufficient epidemiological evidence for causal relationships between several adverse pregnancy or child health outcomes and prenatal or childhood exposure to environmental chemical contaminants. These included prenatal high-level methylmercury (CH(3)Hg) exposure (delayed developmental milestones and cognitive, motor, auditory, and visual deficits), high-level prenatal exposure to polychlorinated biphenyls (PCBs), polychlorinated dibenzofurans (PCDFs), and related toxicants (neonatal tooth abnormalities, cognitive and motor deficits), maternal active smoking (delayed conception, preterm birth, fetal growth deficit [FGD] and sudden infant death syndrome [SIDS]) and prenatal environmental tobacco smoke (ETS) exposure (preterm birth), low-level childhood lead exposure (cognitive deficits and renal tubular damage), high-level childhood CH(3)Hg exposure (visual deficits), high-level childhood exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (chloracne), childhood ETS exposure (SIDS, new-onset asthma, increased asthma severity, lung and middle ear infections, and adult breast and lung cancer), childhood exposure to biomass smoke (lung infections), and childhood exposure to outdoor air pollutants (increased asthma severity). Evidence for some proven relationships came from investigation of relatively small numbers of children with high-dose prenatal or early childhood exposures, e.g., CH(3)Hg poisoning episodes in Japan and Iraq. In contrast, consensus on a causal relationship between incident asthma and ETS exposure came only recently after many studies and prolonged debate. There were many relationships supported by limited epidemiologic evidence, ranging from several studies with fairly consistent findings and evidence of dose-response relationships to those where 20 or more studies provided inconsistent or otherwise less than convincing evidence of an association. The latter included childhood cancer and parental or childhood exposures to pesticides. In most cases, relationships supported by inadequate epidemiologic evidence reflect scarcity of evidence as opposed to strong evidence of no effect. This summary points to three main needs: (1) Where relationships between child health and environmental exposures are supported by sufficient evidence of causal relationships, there is a need for (a) policies and programs to minimize population exposures and (b) population-based biomonitoring to track exposure levels, i.e., through ongoing or periodic surveys with measurements of contaminant levels in blood, urine and other samples. (2) For relationships supported by limited evidence, there is a need for targeted research and policy options ranging from ongoing evaluation of evidence to proactive actions. (3) There is a great need for population-based, multidisciplinary and collaborative research on the many relationships supported by inadequate evidence, as these represent major knowledge gaps. Expert groups faced with evaluating epidemiologic evidence of potential causal relationships repeatedly encounter problems in summarizing the available data. A major driver for undertaking such summaries is the need to compensate for the limited sample sizes of individual epidemiologic studies. Sample size limitations are major obstacles to exploration of prenatal, paternal, and childhood exposures during specific time windows, exposure intensity, exposure-exposure or exposure-gene interactions, and relatively rare health outcomes such as childhood cancer. Such research needs call for investments in research infrastructure, including human resources and methods development (standardized protocols, biomarker research, validated exposure metrics, reference analytic laboratories). These are needed to generate research findings that can be compared and subjected to pooled analyses aimed at knowledge synthesis.

Source: Medline

8. Does secondary smoke exposure increase the incidence and/or severity of asthma in children?

Author(s) Arshad M, Hamm RM, Mold JW

Citation: Journal - Oklahoma State Medical Association, February 2006, vol./is. 99/2(76-7), 0030-1876;0030-1876 (2006 Feb)

Publication Date: February 2006
Abstract: Pediatric asthma is a significant health problem in the United States. Up to 26,000 new asthma cases are identified every year. Seventeen percent of all pediatric emergency department visits are attributable to asthma. There are no universally agreed upon diagnostic criteria for asthma. Because no single agent has been identified as causing asthma and because no pathologic feature is entirely unique to asthma, the disease can more easily be described than defined. Asthma is diagnosed clinically based upon recurrent episodes of wheezing, breathlessness, chest tightness and coughing, particularly at night in the absence of other causes. Asthma is considered a chronic inflammatory disorder associated with airflow obstruction, which is often reversible either spontaneously or with treatment. This inflammation exacerbates bronchial hyper-responsiveness to a variety of environmental stimuli including allergens and irritants. Due to inconsistency of diagnostic criteria for asthma, it is easier to measure asthma severity or to study events such as hospitalizations or deaths, rather than to measure incidence. Since a randomized controlled trial of the effect of cigarette exposure on asthma would be unethical, we must rely on either randomized trials of reduction of cigarette exposure or epidemiological studies to determine associations between secondary exposure to cigarette smoke and asthma.

Source: Medline


Author(s) Halken S

Citation: Pediatric Allergy & Immunology, June 2004, vol./is. 15 Suppl 16/(4-5, 9-32), 0905-6157;0905-6157 (2004 Jun)

Publication Date: June 2004

Abstract: The development and phenotypic expression of atopic diseases depends on a complex interaction between genetic factors, environmental exposure to allergens, and non-specific adjuvant factors, such as tobacco smoke, air pollution and infections. Preventive measures may include both exposure to allergens and adjuvant risk/protective factors and pharmacological treatment. These measures may address the general population, children at risk for development of atopic disease (high-risk infants), children with early symptoms of allergic disease or children with chronic disease. The objective for this review was to evaluate possible preventive measures as regards prevention of development of allergic disease in childhood--primary prevention--and also some aspects of the effect of specific allergy treatment as regards secondary prevention in children with allergic asthma and allergic rhinoconjunctivitis. In one prospective observational study of a birth cohort of unselected infants we evaluated possible predictive/risk factors. In two prospective intervention studies including 1 yr birth cohorts of high-risk (HR) infants we investigated the effect of feeding HR infants exclusively breast milk (BM) and/or hydroyzed cow's milk-based formula the first 4-6 months as regards: (i) the allergy preventive effect of BM/extensively hydrolysed formula (eHF) compared with ordinary cow's milk-based formula, (ii) the effect of two different eHFs, a whey (Profylac) and a casein-based (Nutramigen) formula, as regards development of cow's milk protein allergy (CMA), and (iii) a comparison of the preventive effect of eHF (Profylac/Nutramigen) with a partially hydrolyzed cow's milk-based formula (pHF) (NanHA) as regards development of CMA. None of the mothers had a restricted diet during pregnancy or lactation period. In two prospective randomized intervention studies we evaluated the preventive effect of specific allergen avoidance and specific immunotherapy (SIT) in children with allergic asthma and allergic rhinoconjunctivitis, respectively. The combination of atopic heredity and elevated cord blood IgE resulted in the best predictive discrimination as regards development of allergic disease. The optimal high-risk group was defined by either double parental atopic predisposition or single atopic predisposition, the latter combined with a cord blood IgE > or = 0.3 kU/1. 66% of unselected infants were daily exposed to tobacco smoke, which was a significant risk factor for recurrent wheezing until the age of 1.5 yr. HR infants were breastfed for a longer period and less exposed to tobacco smoke than unselected infants. Exclusively BM/eHF for at least 4 months was associated with a significantly reduced cumulative prevalence of CMA [3.6% (5/141) vs. 20% (15/75) in the control group] up to 5 yr. The effect of the two different eHFs was similar. Exclusively breastfed infants were significantly less exposed to tobacco smoke and pets, had solid foods introduced later and belonged to higher social classes. pHF was significantly (p = 0.05) less effective than eHF
as regards prevention of development of CMA. A diet period of 4 months seems to be as efficient as 6 months or more as regards development of CMA. A few ongoing prospective, randomized intervention studies have produced the first indication that avoidance of indoor allergens such as house dust mite (HDM) in HR infants may reduce the incidence of severe wheeze and sensitization during the first 1-4 yr of age. Long-term follow-up is awaited. In a prospective, double-blind placebo-controlled study in children with doctors diagnosed asthma and documented HDM allergy, we found that semipermeable polyurethane mattress and pillow encasings (Allergy Control) when compared with placebo encasings resulted in a significant perennial reduction of HDM exposure and a significant reduction in the needed dose of inhaled steroids by approximately 50% (mean dose: 408 microg--227 microg/day) after 1-yr follow-up. In another randomized prospective study we investigated the possible preventive effect of SIT in children with allergic rhinoconjunctivitis and grass/birch pollen allergy as regards development of asthma. Among those without asthma significantly fewer in the SIT group developed asthma when compared with the control group (19/79 = 24% vs.32/72 = 44%) after the first 3 yr; and methacholinebronchial provocation test results improved significant in the SIT group. The results of our studies support the evidence that the risk for development of early allergic manifestations e.g. CMA and atopic dermatitis can be reduced significantly by simple dietary measures for the first 4 months of life. In all infants breastfeeding should be encouraged for at least 4-6 months, and exposure to tobacco smoke should be avoided during pregnancy and early childhood. In HR infants a documented hypoallergenic formula (at present eHF) is recommended if exclusive breastfeeding is not possible for the first 4 months. In homes of HR-infants, current evidence supports measures to reduce the levels of indoor allergens e.g. HDM and pets. In symptomatic children allergen-specific treatment may influence both the symptoms and the prognosis. Allergen avoidance can reduce the need for pharmacological treatment, SIT may have the potential for preventing the development of asthma in children with allergic rhinoconjunctivitis. and it may be possible to interfere with the natural course of allergic diseases.

Source: Medline

Available in fulltext from Pediatric Allergy and Immunology at EBSCOhost


Author(s) Rushton L, Courage C, Green E

Citation: Journal of the Royal Society for the Promotion of Health, 01 September 2003, vol./is. 123/3(175-180), 14664240

Publication Date: 01 September 2003

Abstract: In this paper, the population attributable risk (PAR), a measure of the excess risk of disease associated with a risk factor, is calculated for some of the common adverse health effects that have been associated with exposure of children to environmental tobacco smoke (ETS): childhood lower respiratory illness, chronic middle ear disease, asthma and sudden infant death syndrome (SIDS). Published data on both risk estimates and the percentage of children exposed to ETS in the home (prevalence of ETS) have been utilised. The percentage of childhood lower respiratory illness and middle ear disease typically attributable to ETS from either parent smoking ranged from 9′ for asthma prevalence and for referral for glue ear, to 257. for hospital admission for lower respiratory illness. Where data were available to calculate PARs separately for mother only smoking and father only smoking, the PARs were generally larger for mothers only smoking, due mainly to higher odds ratios for mothers only smoking. The PAR for SIDS attributable to ETS from mother only smoking was 11%. Although based on a small number of studies, the PAR for SIDS attributable to smoking of fathers only was similar to that attributable to the smoking of mothers only, largely due to the higher prevalence of households where only the father smokes. This study has shown that the impact of ETS on childhood illness can be considerable, emphasising the importance of the need to develop effective strategies for reducing the risk of ETS exposure in the home and elsewhere.

Source: CINAHL

Author(s): Heudorf U.

Citation: Tagliche Praxis, 2001, vol./is. 42/3(679-689), 0494-464X (2001)

Abstract: Environmental tobacco smoke is the single most important avoidable <<indoor air pollution>>, causing mortality and morbidity in children exposed. Here, reviews and metaanalyses of hundreds of studies with many thousands of children are cited and summarized. Overall there is a very consistent picture with odds ratios for respiratory diseases and middle ear diseases between 1,2 and 1,6 for either parent smoking. Usually odds ratios are higher in preschool children than in school aged children and they are higher when exposed to maternal than to paternal environmental tobacco smoke. For sudden infant death syndrome odds ratio for parental smoking is about 2,0. According to WHO risk assessment up 26% of lower airways diseases in childhood are associated with passive smoking and thus avoidable: in Europe an amount of about 300000 to 500000 diseases per year is estimated. Environmental tobacco smoke is <<environmental pollution no 1>> and poses an important public health problem. The data reported here should help physicians and pediatricians to motivate and persuade parents not to smoke in presence of their children or quit smoking if possible even before pregnancy.

Source: EMBASE


Author(s): Manuel J

Citation: Environmental Health Perspectives, April 1999, vol./is. 107/4(A196-201), 0091-6765;0091-6765 (1999 Apr)

Abstract: One study after another is finding strong associations between a variety of human illness and exposure to environmental tobacco smoke (ETS). A 1986 report by the U.S. Surgeon General concluded that ETS is a cause of disease, including lung cancer, in healthy nonsmokers. Other reports have documented causal associations between ETS and lower respiratory tract infections, middle ear disease and exacerbation of asthma in children, heart disease, retardation of fetal growth, sudden infant death syndrome, and nasal sinus cancer. However, the findings from many of these studies remain controversial. A number of scientists remain skeptical about the association between ETS and serious illness in nonsmokers, charging that scientific journals either fail to publish pro-tobacco findings and meta-analyses or disregard those that are published. They also claim that many epidemiological studies declare causal associations based on marginal odds ratios.

Source: Medline

Available in fulltext from Environmental Health Perspectives at EBSCOhost

Available in fulltext from Environmental Health Perspectives at EBSCOhost

Available in fulltext from Environmental Health Perspectives at National Library of Medicine

13. Health effects of passive smoking-10: Summary of effects of parental smoking on the respiratory health of children and implications for research.

Author(s): Cook DG, Strachan DP

Citation: Thorax, April 1999, vol./is. 54/4(357-66), 0040-6376;0040-6376 (1999 Apr)

Abstract: BACKGROUND: Two recent reviews have assessed the effect of parental smoking on respiratory disease in children.METHODS: The results of the systematic quantitative review published as a series in Thorax are summarised and brought up to date
by considering papers appearing on Embase or Medline up to June 1998. The findings are compared with those of the review published recently by the Californian Environmental Protection Agency (EPA). Areas requiring further research are identified.

RESULTS: Overall there is a very consistent picture with odds ratios for respiratory illnesses and symptoms and middle ear disease of between 1.2 and 1.6 for either parent smoking, the odds usually being higher in pre-school than in school aged children. For sudden infant death syndrome the odds ratio for maternal smoking is about 2. Significant effects from paternal smoking suggest a role for postnatal exposure to environmental tobacco smoke. Recent publications do not lead us to alter the conclusions of our earlier reviews. While essentially narrative rather than systematic and quantitative, the findings of the Californian EPA review are broadly similar. In addition they have reviewed studies of the effects of environmental tobacco smoke on children with cystic fibrosis and conclude from the limited evidence that there is a strong case for a relationship between parental smoking and admissions to hospital. They also review data from adults of the effects of acute exposure to environmental tobacco smoke under laboratory conditions which suggest acute effects on spirometric parameters rather than on bronchial hyperresponsiveness. It seems likely that such effects are also present in children.

CONCLUSIONS: Substantial benefits to children would arise if parents stopped smoking after birth, even if the mother smoked during pregnancy. Policies need to be developed which reduce smoking amongst parents and protect infants and young children from exposure to environmental tobacco smoke. The weight of evidence is such that new prevalence studies are no longer justified. What are needed are studies which allow comparison of the effects of critical periods of exposure to cigarette smoke, particularly in utero, early infancy, and later childhood. Where longitudinal studies are carried out they should be analysed to look at the way in which changes in exposure are related to changes in outcome. Better still would be studies demonstrating reversibility of adverse effects, especially in asthmatic subjects or children with cystic fibrosis.

Source: Medline
Available in fulltext from Thorax at Highwire Press
Available in fulltext from Thorax at National Library of Medicine
Available in fulltext from Thorax at Highwire Press


Author(s) Strachan DP, Cook DG
Citation: Thorax, March 1998, vol./is. 53/3(204-12), 0040-6376;0040-6376 (1998 Mar)
PUBLICATION Date: March 1998

Abstract: BACKGROUND: The relation of parental smoking to wheezing and asthma occurring after the first year of life was assessed by a systematic quantitative review of case-control and longitudinal studies, complementing earlier reviews of cross sectional surveys and wheezing in early childhood.METHODS: Fifty one relevant publications were identified after consideration of 1593 abstracts selected by electronic search of the Embase and Medline databases using keywords relevant to passive smoking in children. The search was completed in April 1997 and identified six studies of asthma incidence, seven of prognosis, 22 case-control studies, and 10 case series addressing disease severity.RESULTS: Maternal smoking was associated with an increased incidence of wheezing illness up to age 6 (pooled odds ratio 1.31, 95% CI 1.22 to 1.41), but less strongly thereafter (1.13, 95% CI 1.04 to 1.22). The long term prognosis of early wheezing illness was better if the mother smoked. The pooled odds ratio for asthma prevalence from 14 case-control studies was 1.37 (95% CI 1.15 to 1.64) if either parent smoked. Four studies suggest that parental smoking is more strongly associated with wheezing among non-atopic children. Indicators of disease severity including symptom scores, attack frequency, medication use, hospital attendance, and life threatening bronchospasm were in general positively related to household smoke exposure.CONCLUSIONS: The excess incidence of wheezing in smoking households appears to be largely non-atopic "wheezy bronchitis" with a relatively benign prognosis, but among children with established asthma, parental smoking is associated with more severe disease. This apparent paradox may be reconciled if environmental tobacco smoke is considered a co-factor provoking wheezing
attacks, rather than a cause of the underlying asthmatic tendency.

Source: Medline
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15. Tobacco and children. An economic evaluation of the medical effects of parental smoking.

Author(s) Aligne CA, Stoddard JJ

Citation: Archives of Pediatrics & Adolescent Medicine, July 1997, vol./is. 151/7(648-53), 1072-4710;1072-4710 (1997 Jul)

Publication Date: July 1997

Abstract: OBJECTIVE: To determine the economic influence of pediatric disease attributable to parental smoking. DATA SOURCES: Computerized bibliographic databases were searched. Subject headings included asthma, burn, cost, low birth weight, otitis media, respiratory syncytial virus bronchiolitis, sudden infant death syndrome, and tobacco smoke pollution. The following constraints were applied to the published articles we studied: publication time, January 1980 through May 1996; age range of children studied, neonate to 18 years; and written in English. Articles used specifically as references for cost issues were limited to studies performed in the United States. DATA EXTRACTION: This study is a literature synthesis, which uses as its primary source the results of previously published best estimates. This is not a meta-analysis of studies analyzing the relationships between childhood disease and smoking. RESULTS: Using data for relative risk, prevalence, and cost of illness and death, we calculated the attributable risk fraction and corresponding direct medical expenditures and costs for loss of life. Costs are adjusted to 1993 dollars. Estimated annual excess cases of childhood illness and death attributable to parental smoking include low birth weight (46,000 cases, 2800 perinatal deaths), sudden infant death syndrome (2000 deaths), respiratory syncytial virus bronchiolitis (22,000 hospitalizations, 1100 deaths), acute otitis media (3.4 million outpatient visits), otitis media with effusion (110,000 tympanostomies), asthma (1.8 million outpatient visits, 14 deaths), and fire-related injuries (10,000 outpatient visits, 590 hospitalizations, and 250 deaths). CONCLUSIONS: Parental smoking is an important preventable cause of morbidity and mortality among American children; it results in annual direct medical expenditures of $4.6 billion and loss of life costs of $8.2 billion. Additional efforts to reduce children's exposure to tobacco smoke are warranted.

Source: Medline


Author(s) Bascom R

Citation: Toxicology Letters, August 1996, vol./is. 86/2-3(115-30), 0378-4274;0378-4274 (1996 Aug)

Publication Date: August 1996

Abstract: Diverse environmental exposure profiles exist in the Americas because of widely different climates, ambient pollutants, and bioaerosols in these continents. This paper reviews selected studies from the Americas that support the broad hypothesis that environmental factors contribute to respiratory hypersensitivity. Processes influenced by environmental factors include primary immunologic sensitization, the development and exacerbation of specific immunologic diseases and the activation of nonspecific mechanisms with tissue inflammation, injury and remodeling. Endpoints resulting from these processes include respiratory symptoms, diseases such as asthma, with measures of disease severity including medication use and hospitalization rates, and death due to cardiorespiratory disease. Studies associate sensitization rates to specific allergens with environmental factors such as humidity and indices of allergen exposure. Regional variation occurs with exposure to outdoor source pollutants such as ozone, but varies by
household to bioaerosols such as dust mite, cat or cockroach allergen. Indoor allergens are associated with asthma while outdoor allergens are associated with allergic rhinitis. In a national survey, the atop sensitization rate in the USA increased with urban residence (defined as towns of population > 2500) and varied by region. Controlled human challenge studies show that ozone increases the response of allergic subjects to allergen. Increased ambient photochemical pollution concentrations, of which ozone is an important component, are associated with increased emergency room visits for asthma in cities such as Toronto, New York, Atlanta, and Mexico City. In Sao Paolo, Brazil, mortality due to childhood respiratory disease was influenced by the ambient levels of NO2. Epidemiologic studies including the recent meta-analysis of a large, longitudinal study population associate ambient concentrations of particulate matter < 10 microns and respiratory symptoms, disease severity and increased cardiorespiratory deaths. Toxicology studies show that individual variation in responsiveness is important in nonspecific inflammatory responses to irritant pollutants such as ozone and environmental tobacco smoke. These studies indicate that environmental factors influence primary allergen sensitization, secondary allergic responses, the activation of nonspecific inflammatory responses, and the severity of respiratory diseases, including asthma.

Source: Medline

17. Morbidity and mortality in children associated with the use of tobacco products by other people

Author(s) DiFranza J.R., Lew R.A.

Citation: Pediatrics, April 1996, vol./is. 97/4(560-568), 0031-4005 (April 1996)

Publication Date: April 1996

Abstract: Objective. To evaluate the impact of adult tobacco use on the health of children. Design. A literature review identified relevant research reports. Meta-analysis was used to compute a pooled risk ratio for each condition studied. The risk ratios were combined with data on exposure rates to produce estimates of the population-attributable risk. Results. Each year, among American children, tobacco is associated with an estimated 284 to 360 deaths from lower respiratory tract illnesses and fires initiated by smoking materials, more than 300 fire-related injuries, 354 000 to 2.2 million episodes of otitis media, 5200 to 165 000 tympanostomies, 14 000 to 21 000 tonsillectomies and/or adenoidectomies, 529 000 physician visits for asthma, 1.3 to 2 million visits for coughs, and in children younger than 5 years of age, 260 000 to 436 000 episodes of bronchitis and 115 000 to 190 000 episodes of pneumonia. Conclusions. The use of tobacco products by adults has an enormous adverse impact on the health of children. Although more research is needed in several areas, action to reduce the morbidity and mortality among children should not be delayed. New laws and policies are needed to grant children protection from bodily injury and death attributable to the use of tobacco products by others.

Source: EMBASE

Available in fulltext from Pediatrics at American Academy of Pediatrics

Safeguarding or child protection


Author(s) Polanska K, Hanke W, Ronchetti R, van den Hazel P, Zuurbier M, Koppe JG, Bartonova A

Citation: Acta Paediatrica Supplement, October 2006, vol./is. 95/453(86-92), 0803-5326:0803-5326 (2006 Oct)

Publication Date: October 2006

Abstract: Almost half of the child population is involuntarily exposed to environmental tobacco smoke (ETS). The ETS exposure gives rise to an excessive risk of several diseases in infancy and childhood, including sudden infant death syndrome, upper and lower respiratory infections, asthma and middle ear diseases. It is also linked to cancer, and behavioural problems and neurocognitive deficits in children. CONCLUSIONS: Protecting children from ETS exposure is a complex and important issue. The best
improvement in children's health is to be gained when parents stop smoking or, when that is not possible, they stop smoking in their children's environment. Paediatricians, because of their authority, and their frequent and regular contact with parents, play a leading role in protecting children from ETS exposure. An ideal approach to help parents to stop smoking seems to be initial minimal-contact advice provided by their paediatrician with feedback and supplemental printed materials, leading to greater intensity and duration of follow-up home visits.

Source: Medline
Available in fulltext from Acta Paediatrica. Supplement at EBSCOhost

2. Protecting children from passive smoking

Author(s)

Citation: Medicine Today, June 2004, vol./is. 5/6(7), 1443-430X (June 2004)
Publication Date: June 2004
Source: EMBASE

3. Protecting children: reducing their environmental tobacco smoke exposure.

Author(s) Klerman L

Citation: Nicotine & Tobacco Research, April 2004, vol./is. 6 Suppl 2/(S239-53), 1462-2203;1462-2203 (2004 Apr)
Publication Date: April 2004
Abstract: The present review examines the current status of efforts to reduce environmental tobacco smoke exposure (ETS) among infants and young children. Estimates of the number of children exposed vary, but it is probably over 20 million or about 35% of all U.S. children. Healthy People 2010 sets as an objective the reduction, to 10%, of the proportion of children regularly exposed to tobacco smoke at home. Children with ETS exposure are at higher risk for upper respiratory illnesses, asthma, otitis media, and sudden infant death syndrome. Eight experimental or quasi-experimental studies of attempts to reduce children's ETS exposure with sample sizes of greater than 100 were conducted in the United States and published between 1990 and 2003. Most of these studies showed a significant impact on maternal smoking and on the number of cigarettes smoked in the home, although intervention-control differences were relatively small. Despite support from professional organizations and federal government groups, many pediatricians and family physicians do not routinely engage in intensive efforts to reduce children's ETS exposure. Training in techniques for reducing tobacco dependence should be included in professional education programs. Public and private insurance should reimburse providers for efforts in this area. An overall strategy for reducing children's ETS exposure should combine individual counseling and education in offices, clinics, and homes with community education and regulatory and economic policies (i.e., smoking bans and excise taxes). Additional funding is needed for studies of provider knowledge, attitudes, and practices; of the effectiveness of various communication strategies; and of office- and community-based strategies to reduce ETS exposure.

Source: Medline
Available in fulltext from Nicotine and Tobacco Research at EBSCOhost

4. Protecting children from environmental tobacco smoke (ETS) exposure: A critical review

Author(s) Gehrman C.A., Hovell M.F.

Citation: Nicotine and Tobacco Research, June 2003, vol./is. 5/3(289-301), 1462-2203 (June 2003)
Publication Date: June 2003
Abstract: Environmental tobacco smoke (ETS) is a significant public health burden. This review summarizes empirical evidence for reducing residential ETS exposure in youth in 19
studies published between 1987 and 2002. Most studies have investigated minimal contact, physician office-based interventions, although some have been conducted in homes and have been more intensive. Interventions are compared based on study design, type of intervention, sample characteristics (asthmatic or healthy), goals, and outcomes, including effect sizes. The average effect size (Cohen's $d$) was .34, with a range from -.14 to 1.04. The evidence suggests that interventions can be effective in reducing children's exposure. More rigorous study designs, interventions of greater intensity and duration, and those based on sound behavior change theory have yielded the most promising results. Challenges and limitations in this area of study are examined. A model for ETS interventions is proposed, and directions for future research are set forth.

Source: EMBASE
Available in fulltext from Nicotine and Tobacco Research at EBSCOhost

5. Protecting children in cars from tobacco smoke [4]

Author(s) Bauman A., Chen X.C., Chapman S.
Citation: British Medical Journal, 1995, vol./is. 311/7013(1164), 0959-8146 (1995)
Publication Date: 1995
Source: EMBASE
Available in print at Grantham Hospital Staff Library

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Health effects of passive smoking. 3. Parental smoking and prevalence of respiratory symptoms and asthma in school age children.
DG Cook, DP Strachan - Thorax, 1997 - thorax.bmj.com
... using a "random effects" model since, in a number of instances, there was a positive response to "Has this child ever had ... A few have reported on current evidence of statistically significant heterogeneity of the passive smoking effect between asthma, usually defined as in ...
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Meta-analysis on the association between environmental tobacco smoke (ETS) exposure and the prevalence of lower respiratory tract infection in early childhood
JSM Li, JK Peat, W Xuan, G Berry - Pediatric pulmonology, 1999 - Wiley Online Library
... 55 Jorm L, Blyth F, Chapman S, Reynolds C. Smoking in child family day care homes: Policies and practice in ... Influence of family factors on asthma and wheezing during the first five years of life. ... Passive smoking, gas cooking, and respiratory health of children living in six cities. ...
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β2-Adrenoceptor polymorphisms and asthma phenotypes: interactions with passive smoking
G Zhang, CM Hayden, SK Khoo, P Candelaria… - European Respiratory …, 2007 -
... Significant exposure was defined as the child having lived with at least one β 2 – adrenoceptor gene single nucleotide polymorphisms studied (Arg16Gly and Gln27Glu) and passive smoking in respect ... Association of β 2 -adrenergic receptor polymorphisms with severe asthma. ...

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Passive smoking respiratory risk. A quantitative synthesis of the literature]

... Wheezing and asthma are likewise more frequent among children exposed to ETS ... current state of knowledge on health risks associated with passive smoking warrants that ... Adolescent; Adult; Child; Female; Forced Expiratory Volume; Humans; Lung Diseases/epidemiology; Lung ...
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Linkage between smoking and asthma

A Pietinalho, A Pelkonen, P Rytilä - Allergy, 2009 - Wiley Online Library
... Health effects assessments for environmental tobacco smoke. ... Health effects of passive smoking 7. Parental smoking, bronchial reactivity and peak flow variability in children. ... Effect of maternal smoking during pregnancy and a family history of asthma on respiratory function in ...
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Impact of environmental tobacco smoke and active tobacco smoking on the development and outcomes of asthma and rhinitis

CE Baena-Cagnani, RM Gómez... - Current opinion in ..., 2009 - journals.lww.com
... Secondhand smoke contains at least 250 chemicals known to be toxic, including more than 50 ... A higher prevalence of wheeze and doctor-diagnosed asthma was also found. ... The different effect of active and passive smoking described in this article highlights some controversy ...
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Health impact assessment of environmental tobacco smoke in European children: sudden infant death syndrome and asthma episodes

E Boldo, S Medina, M Öberg, V Puklová... - Public Health ..., 2010 - ncbi.nlm.nih.gov
... a causal relationship between parental smoking and ever having asthma among children ... Passive smoking and sudden infant death syndrome: review of the epidemiological evidence. ... Exposure to secondhand smoke among students aged 13–15 years— worldwide, 2000–2007 ...
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Developing asthma in childhood from exposure to secondhand tobacco smoke: insights from a meta-regression

K Vork, R Broadwin, R Blaisdell - Ciência & Saúde Coletiva, 2008 - SciELO Brasil
... Exposure key words included ETS, environmental tobacco smoke, passive smoking, secondhand smoke, involuntary smoke, tobacco smoke ... between indoor and outdoor air pollution and adolescent asthma from 1995 to ... S, Host A, Nilsson L, Taudorf E. Passive smoking as a ...
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Prenatal and postnatal environmental tobacco smoke exposure and children's health

JR DiFranza, CA Aline, M Weitzman - Pediatrics, 2004 - Am Acad Pediatrics
... recognized since 1957, 1 and the first report concerning the adverse effects of environmental tobacco smoke (ETS) on ... gender, age, urbanization, education, crowding, dampness, mold, cooking fuel, parental respiratory symptoms, parental asthma, and the child's smoking. ...
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Both environmental tobacco smoke and personal smoking is related to asthma and wheeze in teenagers.

L Hedman, A Bjerg, S Sundberg, B Forsberg, ... - Thorax, 2011 - thorax.bmj.com

... Both environmental tobacco smoke and personal smoking is related to asthma and wheeze in teenagers. ... Prevalence (%) of asthma, wheeze, environmental tobacco smoke (ETS) exposure at age 7–8 and 16–17 years, and personal smoking habits at age 16–17 years. ...

Cited by 13 Related articles All 7 versions Cite

The effect of passive smoking on respiratory health in children and adults State of the Art

C Janson - The International Journal of Tuberculosis and Lung ..., 2004 - ingentaconnect.com

... 32 Larsson ML, Frisk M, Hallstrom J, Kiviloog J, Lundback B. Environmental tobacco smoke exposure during childhood is associated with increased prevalence of asthma in adults. ... 8. Passive smoking and risk of adult asthma and COPD: an update. ...

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Adverse health effects of prenatal and postnatal tobacco smoke exposure on children

W Hofhuis, JC De Jongste, P Merkus - Archives of disease in ..., 2003 - adc.bmj.com

... The adverse effects of passive smoking on the health of the fetus and child are thought to ... the number of Dutch infants (0–1 year of age) exposed to environmental tobacco smoke (ETS) is ... an easy delivery, they are unaware of the possible life-long consequences for their child. ...

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... by 1988, on the matter of respiratory health effects of environmental tobacco smoke exposure (ETS) ... Wheezing and asthma are likewise more frequent among children exposed to ETS at ... the current state of knowledge on health risks associated with passive smoking warrants that ...

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Application of Neuman's framework: Infant exposure to environmental tobacco smoke

MBF Stepans, JR Knight - Nursing Science Quarterly, 2002 - nsq.sagepub.com

... Using Neuman's Systems Framework NOTE: CO = carbon monoxide; ETS = environmental tobacco smoke; SIDS = Sudden ... association between air pollution and the development of asthma is controversial ... Passive smoking and middle ear effusion among children in day care ...

Cited by 7 Related articles All 4 versions Cite

Parental smoking and respiratory tract infections in children

JK Peat, V Keena, Z Harakeh, G Marks - Paediatric respiratory reviews, 2001 - Elsevier

... Effects of environment and passive smoking on the respiratory health of children. ... Prevalence and severity of childhood asthma and allergic sensitisation in seven climatic regions of ... The burden of environmental tobacco smoke exposure on the respiratory health of children 2 ...

Cited by 48 Related articles All 6 versions Cite

Health effects of passive smoking. 1. Parental smoking and lower respiratory illness in infancy and early childhood.

DP Strachan, DG Cook - Thorax, 1997 - thorax.bmj.com

... Studies of asthma incidence, hospital studies are broadly consistent, prognosis, and severity will be reviewed ... were: piratory illness in infancy is very likely (1) To identify all passive smoking ref- to ... each (b) search (c) above for paediatric$ or pediatric$ or infan$ or child$ or study ...

Cited by 359 Related articles All 8 versions Cite
Safeguarding

Protecting sick children from exposure to passive smoking through mothers’ actions: a randomized controlled trial of a nursing intervention

S Chan, TH Lam - Journal of advanced nursing, 2006 - Wiley Online Library
... included a brief proposal of the study, the standardized health advice script, the booklets, the questionnaires, and a resource book on ‘passive smoking’. ... Table 1. Environmental tobacco smoke and health education study – health advice script. Nurse greets the child’s mother. ...
Cited by 27 Related articles All 5 versions Cite

Reducing children's exposure to environmental tobacco smoke in homes: issues and strategies

MJ Ashley, R Ferrence - Tobacco Control, 1998 - tobaccocontrol.bmj.com
... Programmes and policies to reduce exposure to environmental tobacco smoke (ETS) in public places and ... In the United States and Canada, children with asthma have been the focus of ... to address ETS control in homes is not known, although child protection legislation could be ...
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Parental smoking cessation to protect young children: a systematic review and meta-analysis

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Reducing secondhand smoke exposure among children and adolescents: emerging issues for intervening with medically at-risk youth

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